EJA

Eur J Anaesthesiol 2023; **40:**495–500

ORIGINAL ARTICLE

Alveolar target ventilation and dead space in children under anaesthesia

The proventiped cohort study

Aurelien Gardon, Mathilde De Queiroz Siqueira, Eloise Cerceuil, Noureddine Bouhamri, Dominique Chassard and Florent Baudin

INTRODUCTION Ventilator settings in children under anaesthesia remain difficult because of the changes in the physiology and the high dead space.

OBJECTIVE To determine the alveolar minute-volume to sustain normocapnia in children under mechanical ventilation.

DESIGN A prospective observational study.

SETTINGS This study was performed between May and October 2019 in a tertiary care children's hospital.

PATIENTS Children between 2 months and 12 years, weighing between 5 and 40 kg, admitted for general anaesthesia.

INTERVENTION Volumetric capnography was used to estimate the alveolar and dead space volume (Vd).

MAIN OUTCOME MEASURES Total and alveolar minute ventilation in (ml kg^{-1} min^{-1}) over 100 breaths.

RESULTS Sixty patients were included comprising 20 per group: 5 to 10 kg (group 1), 10 to 20 kg (group 2), 20 to 40 kg (group 3). Seven patients were excluded for aberrant capnographic curves. After normalisation to weight, the median [IQR] tidal volume per kilogram was similar between the three groups: 6.5 ml kg^{-1} [6.0 to 7.5 ml kg^{-1}], 6.4 ml kg^{-1} [5.7 to 7.3 ml kg^{-1}], 6.4 ml kg^{-1} [5.3 to 6.8 ml kg^{-1}]; $P = 0.3$. Total Vd (in ml kg^{-1}) was negatively correlated to weight ($r = -0.62$, 95% confidence interval -0.41 to -0.76, $P < 0.001$). The total normalised minute ventilation (ml kg^{-1} min^{-1}) to obtain normocapnia was higher in group 1 than in group 2 and in group 3; 203 ml kg^{-1} min^{-1} [175 to 219 ml kg^{-1} min^{-1}], 150 ml kg^{-1} min^{-1} [139 to 181 ml kg^{-1} min^{-1}] and 128 ml kg^{-1} min^{-1} [107 to 157 ml kg^{-1} min^{-1}]; $P < 0.001$ (mean ± SD), but (mean ± SD) alveolar minute ventilation was similar between the three groups; 68 ± 21 ml kg^{-1} min^{-1}.

CONCLUSION Total dead space volume (including apparatus dead space) represents a major component of tidal volume in children less than 30 kg, when using large heat and moisture exchanger filters. The total minute ventilation necessary to achieve normocapnia decreased with increasing weight, while the alveolar minute ventilation remained constant.

TRIAL REGISTRATION ClinicalTrials.gov, identifier: NCT03901599

Published online 12 April 2023

KEY POINT

- Total dead space volume is higher than alveolar tidal volume in children less than 30 kg, when using large heat and moisture exchanger filters relative to the child's body weight.
- Total dead space decreases with weight resulting in a decrease of total minute ventilation normalised to body weight required to maintain normocapnia.
- Alveolar ventilation remains constant and may be a target to set the ventilator in children independently of the dead space.
- Limiting the instrumental dead space (i.e. filters) is essential in small infants.

From the Hospices Civils de Lyon, Departement d'anesthesie, Hôpital FemmeMère Enfant, Bron (AG, MDQS, EC, NB, FB, DC), Agressions Pulmonaires et Circulatoires dans le Sepsis (APCSe), VetAgro Sup, Universités de Lyon, Marcy l'Etoile, France (FB)

Correspondence to Florent Baudin, Département d'anesthésie-réanimation pédiatrique, Hôpital Femme Mère Enfant, Hospices Civils de Lyon, 59 Bd Pinel, F-69500 Bron, France
Tel: +33 4 72 12 97 35; fax: +33 4 27 86 92 70; e-mail: florent.baudin@chu-lyon.fr

0265-0215 Copyright © 2023 European Society of Anaesthesiology and Intensive Care. Unauthorized reproduction of this article is prohibited.

DOI:10.1097/EJA.0000000000001832

Copyright © 2023 European Society of Anaesthesiology and Intensive Care. Unauthorized reproduction of this article is prohibited.

Introduction

Dead space corresponds to the volume of inhaled gas that does not participate in gas exchange, while alveolar ventilation is the physiological component of ventilation where gas exchange occurs. The anatomic dead space is proportionally bigger in infants, reaching more than 3 ml kg^{-1} in neonates.[1] During anaesthesia, both the anatomic dead space and the apparatus dead space (filter, tracheal tube and connectors) have to be taken into account,[2] and any increase in the apparatus dead space volume can significantly increase the total dead space to tidal volume ratio (Vd/Vt), potentially leading to hypoventilation in children.[3]

Recommendations regarding the settings of tidal volume in the context of paediatric anaesthesia are lacking and mainly based on studies including adult patients.[4,5] However, as the Vd/Vt ratio is different in children compared with adults, it may be not relevant to recommend the same ideal target tidal volume (6 to 8 ml kg^{-1}) for children as in adults.[6] Moreover, there are physiological differences between children and adults, such as their increased oxygen consumption and CO_2 production due to a higher metabolism on a weight basis,[7,8] and the risk of ventilator-induced lung injury (VILI) in a developing lung, which can explain the difficulty of transferring adult results to paediatric populations.[9,10] Consequently, as a significant part of the volume delivered by the ventilator is taken up by anatomical and apparatus dead space that does not take part in gas exchange, the ventilation required in order to maintain normocarbia must be individualised and adjusted to the dead space volume.[3]

Volumetric capnography, represented by the partial pressure of exhaled CO_2 over the volume of one tidal breath, is a noninvasive tool allowing to estimate the dead space volume and alveolar volume.[11,12] The aim of this study was to determine a target of alveolar minute volume to ensure normocapnia in children under mechanical ventilation for general anaesthesia.

Materials and methods

This prospective observational study was conducted in the paediatric anaesthesia department of a tertiary university hospital [Hôpital Femme Mère Enfant (HFME), Hospices Civils de Lyon, France]. The study protocol was approved by an ethics review board (CPP Ile de France V - no. 19.03.05.41013 at Saint Antoine Hospital, Paris, France on 23 April 2019) and was registered on clinicaltrials.gov (NCT03901599). The manuscript is in accordance with the STROBE (STrengthening the Reporting of OBservational studies in Epidemiology) statement.

Patients

The inclusion criteria were age of 2 months to 12 years, weighing between 5 and 40 kg, admission for general anaesthesia and the parent's (or legal guardian's) informed consent (nonopposition). The exclusion criteria were having a history of bronchodysplasia or laryngo tracheo malacia, cyanotic heart disease, neuromuscular disease, bronchospasm requiring Beta 2-agonists at induction or air leaks more than 10% at the time of recording. Children were divided into three groups depending on their weight: group 1, 5 to 10 kg; group 2, to 20 kg; and group 3, 20 to 40 kg.

Ventilator and respiratory parameters

A paediatric circuit (15 mm, 2 m; Int'air Medical, Bourg-en-Bresse, France) with heat and moisture exchanger (HME) and bacterial-viral filter (volume 13 ml; HumidVent, Gibeck Teleflex, Le Faget, France) were used for children weighing 25 kg or less and adult circuit (22 mm, 1.60 m; Int'air Medical) with HME filter (volume 51 ml, DAR Adult – Pediatric Electrostatic Filter HME /Small, Covidien – Medtronic, Paris, France) were used for children weighing more than 25 kg. Automatic compensation for compressible volume was used for all patients and calibrated according to the circuit and filter size (Ventilator Primus, Dräger, Anthony, France). A paediatric combined mainstream CO_2 and flow sensor (Capnostat-5; Phillips Healthcare, Suresnes, France) was inserted between the Y piece and the filter to measure the total dead space volume, including the HME dead space volume, and connected to a respiratory profile monitor (NM3, Respironics, Philips Healthcare). The dead space volume of the sensor was less than 4 ml and the expired CO_2 was continuously measured using infrared absorption spectroscopy with a delay time less than 60 ms. The respiratory monitor was calibrated on ambient air for pressure, flow and CO_2 before each use and adjusted for sevoflurane use. According to the manufacturer, the precision of the measurement was $\pm$ 2 mmHg for $EtCO_2$ 40 mmHg or less.

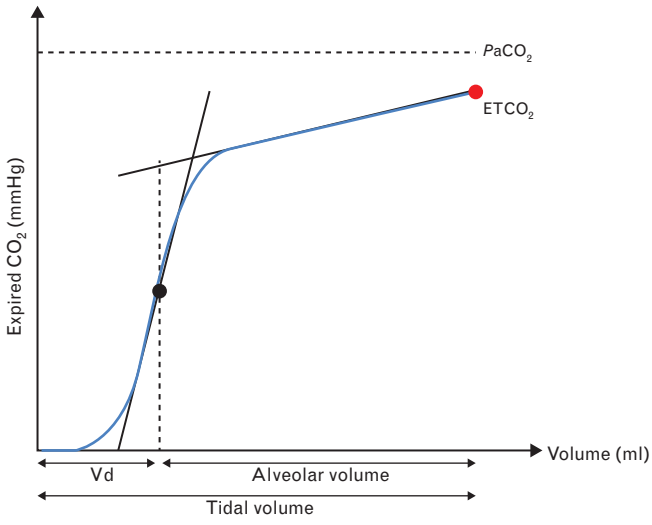
The induction of anaesthesia and the ventilatory mode were at the discretion of the anaesthesiologist. After intubation (cuffed tubes with cuff pressure set at 20 to 25 cmH_2O), children received standard monitoring for general anaesthesia. No recruitment manoeuvre was performed after intubation, but positive end expiratory pressure (PEEP) was used in all children. The respiratory rate was set at 30 min^{-1} in children aged 2 years or less, 25 min^{-1} in children aged 5 years or less and 20 min^{-1} in children aged 12 years or less, and the inspiratory/expiratory time (I/E) ratio between 1/1.5 and 1/2. The tidal volume was initially set (or targeted in pressure control mode) between 6 and 8 ml kg^{-1}; then, ventilatory parameters were adjusted by the anaesthesiologist to obtain normocapnia [defined as an end-tidal CO_2 ($EtCO_2$) between 38 and 42 mmHg].

Data collection

Ventilatory data were collected after a delay of at least 5 min after anaesthesia induction and just before surgery. Baseline demographic data (age, weight and height) were

Copyright © 2023 European Society of Anaesthesiology and Intensive Care. Unauthorized reproduction of this article is prohibited.

Fig. 1 Example of volumetric capnography curve.



ETCO2, end-tidal carbon dioxide; *P*aCO2, arterial partial pressure of carbon dioxide; Vd, dead space volume (i.e. anatomic + apparatus dead space), alveolar volume (i.e. effective alveolar tidal volume and alveolar deadspace).

collected. Data from the NM3 monitor were analysed off-line breath by breath (Fig. 1) and the total dead space volume (airway and apparatus dead space) and alveolar volume were calculated with Fowler's method using the Fletcher's or Bohr's approach as previously described.[11,13–15] Patients with more than 60% aberrant capnographic curves that did not provide an accurate estimation of dead space, mainly due to low tidal volume, were excluded at the time of analysis.[16] The main outcome was the alveolar volume, calculated as the difference between tidal volume and airway dead space, expressed in ml kg^{-1} of real body weight. Alveolar minute ventilation was calculated as the value of the alveolar volume over a one-minute period and expressed in ml kg^{-1} min^{-1}.

Statistical analysis

Categorical data were expressed as count (percentage) and continuous data as median [interquartile range, IQR] or mean ± standard deviation (SD) according to the distribution. Normality was evaluated using the Shapiro–Wilk test. Data were compared between the three groups using a χ^2 test for categorical data and the Kruskall–Wallis test for continuous data. Pairwise comparisons were performed using the Dunn's procedure with a Bonferroni correction for multiple comparisons. A one-way analysis of covariance was performed to compare the main outcome between the three groups adjusted on the end-tidal CO_2 value. Dead space volume and alveolar tidal volume were correlated according to weight using Spearman test.

On the basis of a set tidal volume of 8.7 ± 1.6 ml kg^{-1} in children less than 10 kg,[17] and an estimated alveolar volume of 7 ml kg^{-1} (i.e. physiological airway dead space volume of 1.7 ml kg^{-1}),[1] a sample size of 10 patients per group was estimated to provide a 90% statistical power with a two-sided α level of 5% to detect a difference between the tidal volume and the alveolar volume. To consider potential exclusions at the time of recording or analysis (anticipated based on previous study[16]), and to be more representative, 20 patients per group were included (PASS Sample Size Software; NCSS, Kaysville, Utah, USA). Groups of weight was made in order to ensure a better weight distribution. Statistical analyses were performed using SPSS v28 software (IBM, New York, New York, USA).

Results

Between May 2019 and October 2019, a total of 60 patients were included (20 per group). No patient was excluded at the time of recording for leaks or bronchospasm, but seven patients were excluded at the time of the analysis because of aberrant curves; they all were from the group 1. Hence, a total of 53 patients (13 in group 1, 20 in group 2 and 20 in group 3) were included in the analysis. The median age, weight and height of the three groups are reported in Table 1.

All children, except one, had inhalational induction with sevoflurane 6% and sufentanil and three (6%) children received neuromuscular blockade. The ventilatory mode was the volume control mode for 49 (92%) children and

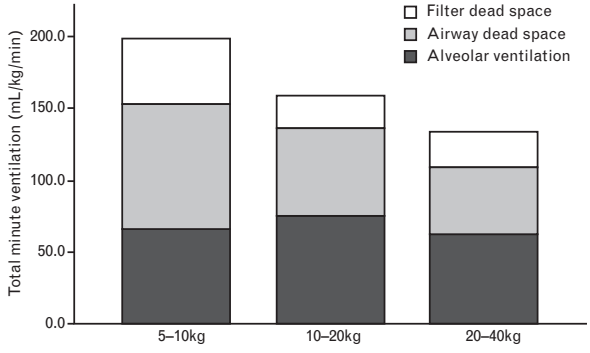
Table 1 Main characteristics of the study population

	Group 1: 5 to 10 kg *n* = 13	Group 2: 10 to 20 kg *n* = 20	Group 3: 20 to 40 kg *n* = 20	*P* value
Weight (kg), median [IQR]	9 [8 to 10]	15 [12 to 17]	29 [22 to 35]	<0.001
Age (months), median [IQR]	12 [8 to 18]	41 [28 to 57]	102 [71 to 122]	<0.001
Height (cm), median [IQR]	74 [68 to 77]	98 [88 to 106]	132 [121 to 139]	<0.001
Anaesthesia duration (min), median [IQR]	106 [90 to 156]	73 [51 to 104]	92 [67 to 120]	0.059
Sex ratio (M/F)	7/6	10/10	8/12	0.82
ASA score, *n* (%)				
ASA 1	7 (54)	12 (60)	12 (60)	0.90
ASA 2	6 (46)	7 (35)	7 (35)	
ASA 3	0	1 (5)	1 (5)	

ASA, American Society of Anaesthesiologists; F, female; IQR, interquartile range; M, male.

Copyright © 2023 European Society of Anaesthesiology and Intensive Care. Unauthorized reproduction of this article is prohibited.

Fig. 2 Total minute ventilation in ml kg^{-1} min according to the weight group.



The airway dead space was calculated by subtracting the filter dead space volume from the total dead space volume. The total minute ventilation was significantly different between groups ($P<0.001$) whereas the minute alveolar ventilation was not ($P=0.16$).

the pressure control mode for four (8%) children. The median total minute ventilation per kg was higher in group 1 than in group 2 and in group 3 [203 ml kg^{-1} min^{-1} (IQR 175 to 219 ml kg^{-1} min^{-1}), 150 ml kg^{-1} min^{-1} (IQR 139 to 181 ml kg^{-1} min^{-1}) and 128 ml kg^{-1} min^{-1} (IQR 107 to 157 ml kg^{-1} min^{-1}); $P<0.001$, Fig. 2] even after adjustment to the $EtCO_2$ value ($P<0.001$). The part of the minute ventilation lost in the filter was higher in group 1 than in group 2 and in group 3 [44 ml kg^{-1} min^{-1} (IQR 40 to 50 ml kg^{-1} min^{-1}), 21 ml kg^{-1} min^{-1} (IQR 19 to 27 ml kg^{-1} min^{-1}) and 23 ml kg^{-1} min^{-1} (IQR 16 to 33 ml kg^{-1} min^{-1}); $P<0.001$, Fig. 2]. However, the alveolar minute ventilation/kg and alveolar minute ventilation/kg adjusted to the $EtCO_2$ value did not differ between the three groups (Table 2 and Fig. 2), and the mean ± SD value was 68 ± 21 ml kg^{-1} min^{-1} for the entire cohort.

The absolute expired median tidal volume not corrected for weight was unsurprisingly different between the three groups [59 ml (IQR 47 to 59 ml), 93 ml (IQR 77 to 111 ml), and 187 ml (IQR 145 to 199 ml) for groups 1, 2 and 3, respectively; $P<0.001$]. The median total dead space volume increased significantly in function of weight [group 1: 36 ml (IQR 34 to 41 ml); group 2: 48 ml (IQR 45 to 53 ml); and group 3: 102 ml (IQR 69 to 115 ml); $P=0.001$], as did the airway dead space, that is without the part due to the filter [group 1: 23 ml (IQR 21 to 28 ml); group 2: 35 ml (IQR 32 to 40 ml); and group 3: 57 ml (IQR 50 to 65 ml); $P<0.001$].

After normalisation against the real body weight, the tidal volume did not differ between the three groups [6.5 ml kg^{-1} (IQR 6.0 to 7.5 ml kg^{-1}), 6.4 ml kg^{-1} (IQR 5.7 to 7.3 ml kg^{-1}) and 6.4 ml kg^{-1} (IQR 5.3 to 6.8 ml kg^{-1}); $P=0.3$], whereas the total dead space/kg remained higher in the group 1 than in the others [4.2 ml kg^{-1} (IQR 4.0 to 4.6 ml kg^{-1}), 3.5 ml kg^{-1} (IQR 3.0 to 3.8 ml kg^{-1}) and 3.2 ml kg^{-1} (IQR 3.0 to 3.6 ml kg^{-1}); $P<0.001$; Table 2]. The median airway dead space, that is without the part due to the filter, was significantly lower in the group 3 than in the others; 2.8 ml kg^{-1} [2.4 to 3.1 ml kg^{-1}], 2.5 ml kg^{-1} [2.2 to 2.8 ml kg^{-1}] and 2.0 ml kg^{-1} 1.7 to 2.4 ml kg^{-1}]; ($P<0.001$; Table 2). Lastly, the dead space volume normalised against the weight (ml kg^{-1}) was negatively correlated with weight [$r=-0.62$ (95% CI -0.41 to -0.76); $P<0.001$], whereas the alveolar tidal volume (ml kg^{-1}) was not significantly correlated with weight [$r=0.23$ (95% CI -0.05 to 0.48); $P=0.10$; Fig. 3].

Discussion

Our study shows that the alveolar minute ventilation did not differ even after adjustment on the end tidal CO_2, while the total minute ventilation normalised on weight decreased with increasing weight. The targeted value of alveolar ventilation was about 70 ml kg^{-1} in the total population in the present study. Interestingly, the part of the tidal volume for the ventilation of the total dead space (including dead space due to the use of large filters) was higher than the alveolar volume for children weighing less than 32.6 kg.

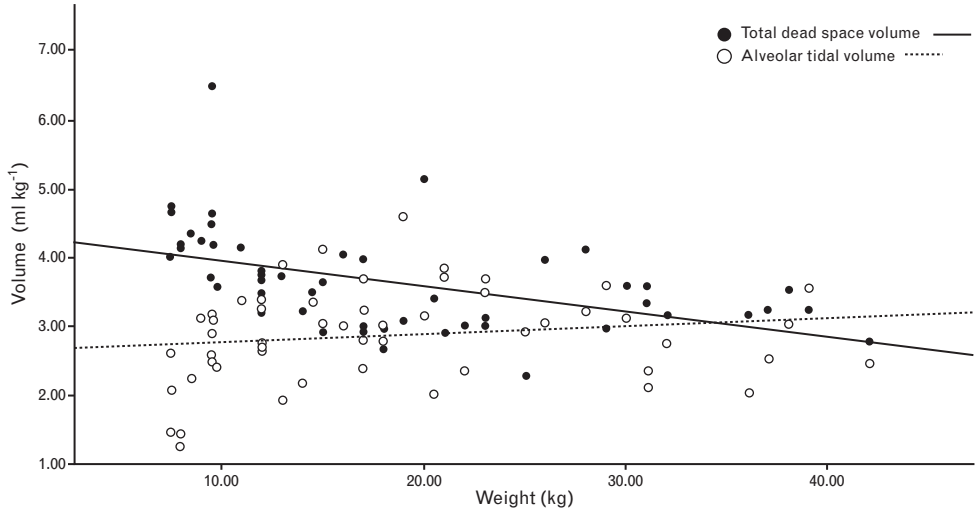
Table 2 Comparison of respiratory and volumetric capnography parameters between the three groups of weight

	Group 1: 5 to 10 kg $n=13$	Group 2: 10 to 20 kg $n=20$	Group 3: 20 to 40 kg $n=20$	*P* value
FiO_2 (%)	40 [40 to 40]	40 [40 to 41]	40 [36–40]	0.067
SpO_2 (%)	99.3 [98.4 to 99.6]	98.9 [97.9 to 99.4]	98.6 [97.5–99.5]	0.63
Heart rate min^{-1}	128 [118 to 138]	112 [100 to 125]	93 [85–106]	<0.001
Respiratory rate min^{-1}	30 [29 to 32]	25 [22 to 26]	21 [18–25]	<0.001
Maximal inspiratory pressure (cmH_2O)	19 [17 to 22]	19 [16 to 21]	18 [16 to 22]	0.61
PEEP (cmH_2O)	4 [3 to 5.3]	4.5 [3.9 to 5.1]	4.5 [4.1 to 5.2]	0.32
EtCO2 (mmHg)	39.0 [36.3 to 40.5]	38.5 [37 to 41]	40.5 [39 to 42]	0.02
Normalised tidal volume (ml kg^{-1})	6.5 [6.0 to 7.5]	6.4 [5.7 to 7.3]	6.4 [5.3 to 6.8]	0.3
Alveolar tidal volume (ml kg^{-1})	2.5 [1.8 to 3.0]	3.0 [2.7 to 3.4]	3.0 [2.4 to 3.5]	0.02
Total dead space (ml kg^{-1})	4.2 [4.0 to 4.6]	3.5 [3.0 to 3.8]	3.2 [3.0 to 3.6]	<0.001
Airway dead space (ml kg^{-1})[a]	2.8 [2.4 to 3.1]	2.5 [2.2 to 2.8]	2.0 [1.7 to 2.4]	<0.001
Alveolar minute ventilation (ml kg^{-1} min^{-1})	69 [47 to 83]	74 [59 to 89]	58 [49 to 75]	0.16
Total minute ventilation (ml kg^{-1} min^{-1})	203 [175 to 219]	150 [139 to 181]	107 [128 to 157]	<0.001

Data are expressed as median [interquartile range]. $EtCO_2$, end-tidal carbon dioxide; FiO_2, fraction of inspired oxygen; PEEP, positive expiratory pressure; SpO_2, pulse oximetry. [a]Airway dead space, total deadspace minus filter deadspace.

Copyright © 2023 European Society of Anaesthesiology and Intensive Care. Unauthorized reproduction of this article is prohibited.

Fig. 3 Total dead space volume in ml kg^{-1} (black point and dotted line) and alveolar volume in ml kg^{-1} (white point and full line) according to weight for each patient.



Total dead space volume was significantly correlated with weight ($P < 0.001$) but not the alveolar tidal volume ($P = 0.10$). The value of weight at the intersection point of the curves was 32.6 kg.

Anatomic dead space is known to be proportionally higher in smaller infants.[1] Numa and Newth[1] have described that the anatomic dead space was 50% greater than in adults and could reach more than 3 ml kg^{-1} in neonates. During anaesthesia, both the anatomic dead space and the apparatus dead space (filter, tracheal tube and connectors) have to be taken into account, and it is of note that herein the high total dead space is mainly explained by the used of large-volume filters relative to the child's bodyweight. As described previously in a mathematical model, the total dead space to tidal volume ratio (Vd/Vt) reaches 0.6 to 0.8 in smallest infants during anaesthesia[2]; our study confirmed this model in real life (value at 0.64 in the smallest group). Total dead space represents, therefore, more than 50% of the tidal volume in children weighing less than 30 kg, which may explain why small children under anaesthesia frequently have to be ventilated with a tidal volume up to more than 10 ml kg^{-1} to achieve normocapnia.[17] This finding has already been reported with similar minute ventilation values.[18] Chau *et al.*[18] have demonstrated that using filters necessitates an increase of 112% in the minute ventilation. However, our study demonstrated that the increase in minute ventilation did not correspond to an increase in alveolar ventilation that remained comparable between the three groups (mean value at 70 ml kg^{-1} min^{-1}). By targeting an alveolar minute ventilation that appears to be constant over the whole population using volumetric capnography, we could ignore the influence of the total dead space volume.

However, it is not possible to ignore the impact of the total dead space volume on the respiratory physiology. Indeed, higher total dead space increases CO_2 retention and may increase arterial partial pressure of carbon dioxide ($PaCO_2$), especially when using low tidal volumes.[19] Even by increasing the minute ventilation, CO_2 remains in the total dead space and therefore becomes mixed with fresh gas in the next inspiratory phase (rebreathing). Moreover, targeting only the minute alveolar ventilation without using physiological parameters (i.e. a very high respiratory rate with a small tidal volume) may lead to an increase of the arterial to end-tidal CO_2 gradient with underestimation of the $PaCO_2$. Indeed, $EtCO_2$ has been shown to be very close to $PaCO_2$ in healthy children, but the difference between $EtCO_2$ and $PaCO_2$ may be higher in infants due to total dead space, ventilation-perfusion mismatch or during laparoscopic surgery.[16,20,21] Hypercarbia may therefore not be fully evaluated when using $EtCO_2$ to assess the effectiveness of ventilation, which also constitutes a limitation of the present study that uses the end-tidal CO_2 as a target, but this constitutes the common approach in the operative room.[4] However, anaesthesiologists should be aware of the risk of underestimating $PaCO_2$ when using $EtCO_2$ in the context of high Vd/Vt ratio. In this case, the use of transcutaneous PCO_2 may be of value, even though the arterial blood gas remains the gold standard.[22]

The present study has several limitations. First, owing to its observational design, there was a variable I/E ratio, heterogeneity in the ventilation mode used; in addition, there was no recruitment manoeuvre with probable subsequent inhomogeneity of alveolar ventilation. However, this reflects current management of children under anaesthesia in our centre, and more widely in France.[17]

Copyright © 2023 European Society of Anaesthesiology and Intensive Care. Unauthorized reproduction of this article is prohibited.

Second, a combined bacterial-viral/HME filter was used in the present study with a dead space volume greater than 10 ml, which represents at least 1 ml kg^{-1} for infants in group 1. The high total dead space volume in the smallest children is influenced by ventilator settings and apparatus dead space, and the results should be interpreted critically, and conclusions adjusted for centres using other settings and filters. To decrease the total dead space, smaller HME filters with a volume of about 3 ml are available and can be used in addition to a bacterial-viral filter connected to the exhalation limb before the ventilator. This option (as the use of an intensive care ventilator with active humidifier) may be preferred for the smallest infants and may, by decreasing the apparatus dead space, improve the accuracy of $EtCO_2$. Third, we excluded one-third of the patients from group 1, but the number of patients included in this group was still sufficient for the analysis (as defined *a priori* in the sample size calculation). However, as alveolar calculation was based on volumetric capnography, the respiratory rate range used in the present study should not have affected the calculation and each curve was review was reviewed individually. As previously described, the volumetric capnography analysed the shape of the curve, and in some patients with small tidal volume the calculation provided aberrant data.[16] This also constitutes a limitation for bedside monitoring because the value provided by the monitor can be inaccurate, especially for small infants. The Fowler method, used to estimate the dead space, does not take into account the alveolar dead space[13]; estimating the alveolar dead space would actually require arterial blood gas and would constitute a major concern for paediatric patients. Moreover, in healthy children, the alveolar dead space remains low compared to the instrumental dead space.

Conclusion

Whereas the total minute ventilation has to be increased to maintain normocapnia in small children, the minute alveolar ventilation per kilogram body weight remains constant over the weight groups and may be a target to set the ventilator, regardless of the Vd/Vt ratio. However, alveolar volume measurement required the use of a volumetric capnography device.

In clinical practice, anaesthesiologists must be aware that total dead space volume represents more than 50% of the tidal volume in children weighing less than 30 kg, especially when using large-volume filters. They should consider this specificity to adjust the ventilator settings in children by avoiding very low tidal volume in the smallest infants, by limiting the instrumental dead space (weight appropriate circuits and HME filters, pre-use self-test of ventilator correctly performed), and by performing a critical appraisal of the $EtCO_2$ value in the context of increased dead space.

Acknowledgements relating to this article

Assistance with the study: the authors thank Hélène Boyer and Philip Robinson (DRS, Hospices Civils de Lyon, France) for help in manuscript preparation. They thank the ALLP, ADENE group for the funding.

Financial support and sponsorship: the study was supported by a grant from the Association Lyonnaise de Logistique Post Hospitalière (ALLP), ADENE group.

Conflict of interest: the authors have no conflict of interest to declare.

Presentation: preliminary data were presented at the French Society of Anaesthesia, Critical Care and perioperative Medecine (SFAR) meeting 2020.

This manuscript was handled by Mona Momeni.

References

1 Numa AH, Newth CJ. Anatomic dead space in infants and children. *J Appl Physiol* 1996; **80**:1485–1489.
2 Pearsall MF, Feldman JM. When does apparatus dead space matter for the pediatric patient? *Anesth Analg* 2014; **118**:1404–1408.
3 King MR, Feldman JM. Optimal management of apparatus dead space in the anesthetized infant. *Pediatr Anesth* 2017; **27**:1185–1192.
4 Feldman JM. Optimal ventilation of the anesthetized pediatric patient. *Anesth Analg* 2015; **120**:165–175.
5 Kneyber MCJ. Intraoperative mechanical ventilation for the pediatric patient. *Best Pract Res Clin Anaesthesiol* 2015; **29**:371–379.
6 Young CC, Harris EM, Vacchiano C, *et al.* Lung-protective ventilation for the surgical patient: international expert panel-based consensus recommendations. *Brit J Anaesth* 2019; **123**:898–913.
7 Brück K. Heat production and temperature regulation. Perinatal physiology. Springer; Boston, MA, USA. pp. 455–98.
8 Lewis RC, Duval AM. Standards for the basal metabolism of children from 2 to 15 years of age, inclusive. *J Pediatrics* 1943; **23**:1–18.
9 Kornecki A, Tsuchida S, Ondiveeran HK, *et al.* Lung development and susceptibility to ventilator-induced lung injury. *Am J Resp Crit Care* 2005; **171**:743–752.
10 Kneyber MCJ, Zhang H, Slutsky AS. Ventilator-induced lung injury. Similarity and differences between children and adults. *Am J Resp Crit Care* 2014; **190**:258–265.
11 Fowler WS. Lung function studies. II. The respiratory dead space. *Am J Physiol* 1948; **154**:405–416.
12 Suarez-Sipmann F, Bohm SH, Tusman G. Volumetric capnography: the time has come. *Curr Opin Crit Care* 2014; **20**:333–339.
13 Bhalla AK, Rubin S, Newth CJL, *et al.* Monitoring dead space in mechanically ventilated children: volumetric capnography versus time-based capnography. *Respir Care* 2015; **60**:1548–1555.
14 Bourgoin P, Baudin F, Brossier D, *et al.* Assessment of Bohr and Enghoff dead space equations in mechanically ventilated children. *Respir Care* 2017; **62**:468–474.
15 Fletcher R, Jonson B, Cummin G, Brew J. The concept of deadspace with special reference to the single breath test for carbon dioxide. *Bja Br J Anaesth* 1981; **53**:77–88.
16 Baudin F, Bourgoin P, Brossier D, *et al.* Noninvasive estimation of arterial CO2 from end-tidal CO2 in mechanically ventilated children. *Pediatr Crit Care Me* 2016; **17**:1117–1123.
17 Lebossé M, Kern D, Queiroz MD, *et al.* Ventilation in pediatric anesthesia: a French multicenter prospective observational study (PEDIAVENT). *Pediatr Anesth* 2020; **30**:912–921.
18 Chau A, Kobe J, Kalyanaraman R, *et al.* Beware the airway filter: deadspace effect in children under 2 years. *Pediatr Anesth* 2006; **16**:932–938.
19 Hinkson CR, Benson MS, Stephens LM, Deem S. The effects of apparatus dead space on P(aCO2) in patients receiving lung-protective ventilation. *Respir Care* 2006; **51**:1140–1144.
20 McSwain SD, Hamel DS, Smith PB, *et al.* End-tidal and arterial carbon dioxide measurements correlate across all levels of physiologic dead space. *Respir Care* 2010; **55**:288–293.
21 Sanders JC, Gerstein N. Arterial to endtidal carbon dioxide gradient during pediatric laparoscopic fundoplication. *Pediatr Anesth* 2008; **18**:1096–1101.
22 Duyu M, Çağlar YM, Karakaya Z, *et al.* Comparison of arterial CO2 estimation by end-tidal and transcutaneous CO2 measurements in intubated children and variability with subject related factors. *J Clin Monitor Comp* 2021; **35**:101–111.

Copyright © 2023 European Society of Anaesthesiology and Intensive Care. Unauthorized reproduction of this article is prohibited.